tient at peak exercise. PkVO2 correlated with percent change in heart rate after anaerobic threshold in groups A (r=0.45, p=0.007) and Bn (r=0.67, p<0.001) only.
Conclusions: Pts with resting LVOTO have poorer indices of cardiac function/exercise capacity. Non-obstructive HCM pts with exercise-induced LVOTO have cardiopulmonary indices consistent with better stroke volume augmentation during exercise.

Table 1. Comparison of exercise parameters between the three groups.

	GROUP A	p(A v Bi)	GROUP Bi	p(Bi v Bn)	GROUP Bn
Peak Workload	62 ±23	<0.001	87 ±21	0.003	67 ±19
pkVO2	65 ±19	<0.001	85 ±16	0.002	68 ±17
pkO2 pulse	82 ±19	0.003	99 ±18	0.004	82 ±19
atVO2	35 ±12	0.007	44 ±13	ns	39 ±9
atO2 pulse	59 ±20	0.008	74 ±18	ns	64 ±15

1015-160 **Quantification of Myocardial Injury After Percutaneous Transluminal Septal Myocardial Ablation in Hypertrophic Obstructive Cardiomyopathy**

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Background: Percutaneous transluminal septal myocardial ablation (PTSMA) is a non-surgical therapeutic procedure for reducing left ventricular outflow tract obstruction in symptomatic patients with hypertrophic obstructive cardiomyopathy (HOCM). The aim of this study was to evaluate septal myocardial injury size after PTSMA using delayed contrast-enhanced (DCE) magnetic resonance imaging (MRI).
Methods: 12 patients (mean age 52±15 years, 7 males) underwent MRI before and 4 weeks after PTSMA; volume of ethanol injected during procedure was 1 to 5 mL. Images were acquired on a 1.5 T scanner (Vision/Sonata, Siemens, Erlangen, Germany). Cine gradient-echo MRI was performed for assessment of global left ventricular function at baseline and follow-up. Inversion-recovery turbo-FLASH images (TE 3.4 ms, TR 7.6 ms, TI 250-300 ms) were acquired at follow-up, 20 to 30 minutes after i.v. administration of 0.2 mmol/kg gadolinium-DTPA. Left ventricular function parameters, myocardial mass, and hyperenhanced area's (including central dark zones of hypoenhancement) were quantified using the MASS software package (Leiden University Medical Center, the Netherlands).
Results: Left ventricular mass values before and after PTSMA were 235.6 ± 70.7 g vs. 225.2 ± 71.7 g (p=0.001), respectively. Septal myocardial mass pre- and post PTSMA were 79.2 ± 29.4 g vs. 72.7 ± 26.2, resp. In all patients the injured myocardium was well visualized. The hyperenhanced septal myocardial mass was in the range of 3.6 to 24.9 g [mean: 12.8 ± 7.8 g], and involved 5.0 % ± 4.0 of the post-ablational total LV mass vs. 21.2 % ± 16.0 of the septal myocardial mass. Myocardial injury size was not correlated with the volume of ethanol administered.
Conclusions: The extent of myocardial injury after PTSMA can be determined using DCE-MRI and was not correlated with the volume of ethanol administered. The method may serve as control and feedback for the interventional procedure.

1015-161 **Untoward Effect of Septal Reduction Interventions on Conduction System in Patients With Obstructive Hypertrophic Cardiomyopathy**

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Hypertrophic obstructive cardiomyopathy (HOCM) patients may develop conduction abnormalities after percutaneous transluminal septal myocardial ablation (PTSMA) and myectomy (MYE) procedures. The aim of the study was to observe the impact of both procedures on the conduction system. **Methods**: A total of 101 HOCM patients without pre-existing permanent pacemaker were enrolled in this study. 55 patients were included in MYE and 46 patients in PTSMA group. Standard 12 leads ECGs were acquired before and 3-month after the procedures. Doppler echo was used to quantify the LVOT pressure gradient (PG). **Results**: Both PTSMA and MYE significantly reduced the LVOT PG (66 ± 41 vs 29 ± 28 mmHg for PTSMA and 68 ± 44 vs 14 ± 10 mmHg for MYE, both p < 0.0001). After PTSMA, a permanent pacemaker was implanted in 13 (28%) patients. 20 patients developed new complete right bundle branch block (CRBBB). After MYE, 6 (11%) patients required permanent pacemakers (p<0.05 vs PTSMA) while 38 patients developed complete left bundle branch block (CLBBB) (table).

	N	Pacer	CRBBB		CLBBB	
			Pre	Post	Pre	Post
PTSMA	46	13 (28%)	3 (7%)	23 (70%)	0 (0%)	2 (2%)
MYE	55	6 (11%)*	0 (0%)	0 (0%)†	3 (5%)	41 (84%)†

MYE vs PTSMA, * p< 0.05, † p < 0.0001.
5 PTSMA patients required subsequent myectomy because of incomplete relief of the PG. 4 of them needed permanent pacemakers. **Conclusion**: Both PTSMA and MYE significantly reduced LVOT PG. However, right branch bundle block was frequently produced by PTSMA and left branch bundle block was often caused by MYE. Severe AV block may develop when both procedures are required.

1015-162 **Prediction of Sudden Death in Patients With Dilated Cardiomyopathy Receiving Angiotensin Converting Enzyme Inhibitors and Beta-Blockers: The Extent of Intercellular Myocardial Fibrosis and the Presence of Nonsustained Ventricular Tachycardia**

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Background: Recent clinical trials have shown that angiotensin-converting enzyme inhibitors (ACEI) and beta-blockers (BB) reduce the mortality in patients with dilated cardiomyopathy (DCM). However, some DCM patients still died suddenly in spite of the administration of ACEI and BB. We tried to identify patients at risk for sudden death in DCM patients receiving ACEI and BB. **Methods:** We studied 50 DCM patients (NYHA class: 2.6±0.6, left ventricular ejection fraction: 32±8%) receiving ACEI (78%) and BB (76%). At the baseline, we performed echocardiography, 24 hour Holter monitoring, right side cardiac catheterization, left ventricular endomyocardial biopsy. The extent of myocardial fibrosis was estimated by the point count method in all biopsy samples. Moreover, after classifying myocardial fibrosis separately into two types, intercellular (IC) and interfascicular fibrosis, by the distribution of fibrosis on each cross point, we also estimated the extent of each fibrosis.
Results: During the observation period of 58±28 months, 8 patients died suddenly. By multivariate Cox analysis, out of the variables including clinical, hemodynamic, echocardiographic parameters, the presence of nonsustained ventricular tachycardia (VT: more than 5 consecutive ventricular ectopic beats) and the extent of myocardial fibrosis, the extent of IC fibrosis (p=0.005) and the presence of nonsustained VT (p=0.04) were independently associated with sudden death. Kaplan-Meier analysis revealed that patients with a greater extent of IC fibrosis (>12.5%) and nonsustained VT (Group I) showed significantly higher rate of sudden death than those with a lesser extent of IC fibrosis or the absence of nonsustained VT (Group II) (67% vs 9%, p<0.0001).
Conclusion: The extent of IC fibrosis and the presence of nonsustained VT would be a powerful predictive marker for sudden death in DCM patients receiving ACEI and BB.

POSTER SESSION

1016 Electrophysiology/Arrhythmias in the Elderly

Sunday, March 17, 2002, 9:00 a.m.-11:00 a.m.
Georgia World Congress Center, Hall G
Presentation Hour: 9:00 a.m.-10:00 a.m.

1016-141 **QT Dispersion Is Not Associated With Left Ventricular Function in Elderly Patients With Symptomatic Heart Failure: ELITE II Substudy**

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Background It remains controversial if QT dispersion (QTd) is associated with left ventricular function or aetiology in patients (pts) with heart failure (HF).
Methods To examine the association between QTd and left ventricular function and the prognostic value of QTd in HF secondary either to idiopathic dilated cardiomyopathy (IDC) or ischaemic heart disease (IHD), we studied 986 HF pts (age 71±7 year, 703 men) enrolled into the ELITE II trial. Of study pts, 768 had IHD (age 71±7 years, 573 men) and 105 had IDC (age 71±7 years, 65 men). QT intervals were manually measured on standard 12-lead ECGs using an in-house computer assisted system.
Results In all study pts, the mean QTd and JTd were 86±31 and 82±32 ms, respectively. In pts with LVEF≤25%, heart rate was significantly higher (P<0.0001) and QT was significantly shorter (p25% (86±31 v 86±31 ms). No significant difference in QTd was found between pts with and without NYHA class III-IV either (87±31 v 84±32 ms). QTd was not correlated with LVEF (r= -0.0004) or NYHA (r= -0.04). During follow-up (540±153 days), 119 (12%) pts died of cardiac cause (CD), including 59 (6%) sudden death (SD). A prospective cut-off value at QTd>80 ms failed to identify pts with CD or SD, while significantly more patients with LVEF≤25% died of CD than those with LVEF>25% (16% v 11%, p=0.045). There were no significant differences in age and NYHA functional class between IHD and IDC. More male (75% v 62%, p=0.006) and higher LVEF (31.6±6.8 v 28.2±7.1%, p<0.001) were found in IHD than IDC. The mean QTd in IHD were similar to IDC (86±32 v 87±27 ms) and it was not related to mortality at all. The frequency of CD or SCD were not significantly different in IDC and IHD (11%, 5% v 13%, 6%, respectively, p=NS). In both IHD and IDC, a QTd >80ms failed to predict CD or SCD. The results remained unchanged when JTd was analysed in the same manner.
Conclusions In this ELITE II substudy, QTd (and JTd) was neither associated with left ventricular function, nor with aetiology of heart failure. Reduced LVEF remains a significant predictor of CD but increased QTd did not predict mortality irrespective of aetiology of HF.